U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 16 August 2007

In the Matter of:

L.R.P.
Claimant,

CASE NO: 2006-BLA-5327

v.

EASTERN ASSOCIATED COAL CORPORATION.,
c/o OLD REPUBLIC INSURANCE COMPANY,
Employer/Carrier,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Derrick W. Lefler, Esq.,
For the Claimant

Paul E. Frampton, Esq.,
For the Employer

Before: LARRY W. PRICE
Administrative Law Judge

DECISION AND ORDER – DENYING BENEFITS

This matter arises from a claim for benefits under the Black Lung Benefits Act, Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. 901 *et seq.* (Act), and applicable Federal Regulation, mainly 20 C.F.R. Parts 412, 718, and 725 (Regulations).

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to coal workers' pneumoconiosis or to the survivors of persons whose death was caused by coal workers' pneumoconiosis. Coal workers' pneumoconiosis is defined

in the Act as "a chronic dust disease of the lung and its sequelae, including pulmonary and respiratory impairments, arising out of coal mine employment." 30 U.S.C. 902(b).

On January 5, 2006, this case was referred to the Office of Administrative Law Judges for a formal hearing. The hearing was held in Pipestem, West Virginia on December 7, 2006. At the trial I admitted Director's exhibits 1 through 27, to the extent that they comply with the evidentiary limitations at 20 C.F.R. § 725.414 (2004); CX[1] 1 and 3; EX 1 through 11. Claimant submitted an interpretation of an x-ray taken in 1999. The x-ray film has since been destroyed by the laboratory, and Employer had not had a chance to have an expert re-read the x-ray film. The regulations specifically address this issue; "the original film on which the X-ray report is based shall be supplied to the Office, unless prohibited by law, in which event the report shall be considered as evidence only if the original film is otherwise available to the Office and other parties." 20 C.F.R. § 718.102(d). Therefore, I will not admit CX 2 into the record. I identified the Claimant's evidence summary form as ALJ 1, and Employer's evidence summary form as ALJ 2. Both parties submit post hearing briefs.

ISSUES

The following issues remain for resolution:

- Existence of pneumoconiosis.
- Whether pneumoconiosis arose from coal mine employment.
- Whether Miner has a totally disabling pulmonary impairment.
- Whether total disability was caused by the pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History

Miner filed the present claim for Black Lung benefits on April 1, 2005. (DX2). On November 8, 2005, a Proposed Decision and Order was issued awarding Claimant benefits under the Act. (DX 21). Employer requested a formal hearing.

Background

Miner was born on November 21, 1943 (DX 2) and currently lives in Bluefield, West Virginia. (Tr. at 19). He married his wife on October 9, 1965; they are still married. (Tr. 19, DX 11). Miner does not have unmarried children under the age of 18, disabled or between 18 and 23 and attending school. (DX 2). Miner has a twelfth grade education. (DX 2). Miner has a coal mine employment history of 34 years.[2] Miner reports working numerous positions in the mines, including clean up man at the preparation plant, mechanic, certified welder and pipe fabricator. His final position in the mines was central control operator. (DX 3).

[1] The following abbreviations have been used in this decision: DX – Director's Exhibit; EX – Employer's Exhibit; CX – Claimant's Exhibit; TR – Transcript of the December 7, 2006 hearing; BCR – Board certified radiologist; B – B-Reader; CWP – Coal Workers' Pneumoconiosis; and COPD – Chronic Obstructive Pulmonary Disease.

[2] Parties stipulated to 34 years of coal mine employment. (Tr. at 37).

The record contains varied statements regarding the Miner's smoking history. Miner testified that he started smoking in his late teens and smokes between one and one and a half packs of cigarettes per day. (Tr. at 27). Dr. Rasmussen reported a smoking history of one and one half packs per day for 44 years. (DX, 13). Dr. Zaldivar's August, 10, 2005 report lists a smoking history of between one and one half packs per day for 43 years. (DX 14). I find that Miner smoked an average of one and one quarter packs of cigarettes a day for 44 years.

MEDICAL EVIDENCE

X-ray Reports

Exhibit	Doctor	Qualifications	Date of X-ray	Date of Reading	Film Quality	Interpretation
CX 1	Robinette	B	8/29/06	10/2/06	1	1/0, q/a
EX 11	Scatarige	B/BCR	8/28/06	12/6/06	Good Quality	No silicosis or CWP; emphysema with probably bullae in upper lobes and left lower lobe; Scarring or plate atelectasis in left lower lobe; atherosclerotic calcification aortic arch.
EX 8	Wheeler	B/BCR	6/14/06	6/27/06	1	No CWP, aa, bu Ill defined 1.5 cm mass or scar left lower lateral lung between anterior ribs. CT scan recommended; could be inflammatory disease or early cancer. Subtle decreased upper lung markings/ check PFTs for emphysema. Few tiny linear scars in left lower lateral lung or bleb walls. Band of discoid atelectasis lower medial right lung.
DX 14	Wheeler	B/BCR	8/10/05	8/23/05	2	No CWP; em, pa; hyperinflation of lungs due to emphysema; few small linear discoid atelectasis or scars lower lingula.
DX 13	Rasmussen[3]	B	6/29/05	7/1/05	1	1/0, s/s; plate atelectasis
DX 14	Scatarige	B/BCR	6/29/05	8/26/05	2	No pneumoconiosis; emphysema and small bullae in the left apex; plate atelectasis in the left lung base

[3] Reread for quality by Dr. Dominic Gaziano. Like Dr. Rasmussen, Dr. Gaziano finds the x-ray film to be of the good quality. (DX 13).

Pulmonary Function Studies[4]

Exhibit #	Physician	Date of Study	Tracings Present?	Flow-Volume Loop?	Broncho-dilator?	FEV1	FVC/ MVV	Age/ Height	Qualify?	Coop and Comp. Noted
DX 13	Rasmussen	6/29/05	Yes	Yes	No	2.63	4.22	61/65	No	Yes
DX 14	Zaldivar	8/10/05	Yes	Yes	B	2.83	4.82	61/66	No	No
					A	2.88	4.71			
EX 5	Zaldivar	6/14/06	Yes	Yes	B	2.81	4.83	62/66	No	No
					A	2.79	4.73			

Arterial Blood Gas Studies[5]

Exhibit #	Physician	Date of Study	Altitude	Resting (R) Exercise (E)	PCO2	PO2	Qualify?	Age	Comments
DX 13	Rasmussen	6/29/05	0-2999	R	33	81	No	61	
				E	33	60	Yes		
DX 14	Zaldivar	8/10/05	0-2999	R	34	87	No	61	
				E	35	68	No		
EX 6	Zaldivar[6]	6/14/06	0-2999	R	26	94	No	62	
				E	30	81	No		

Other Medical Evidence

Exhibit #	Physician	Type of Record	Date of Report	Summary
DX 14	Scott	CT scan[7]	8/10/05	No evidence of silicosis or CWP; bullous emphysema around the periphery of the upper lungs; mild centrilobular emphysema in the u lungs.

[4] 20 C.F.R. 718 Appx. B establishes the standards for the administration and interpretation of pulmonary function tests.

[5] 20 C.F.R. 718 Appx. C establishes the standards for the administration and interpretation of arterial blood gas studies.

[6] There is some question as to whether or not these studies are valid. Miner testified that Dr. Zaldivar stopped the exercise prematurely and only obtained the blood gas samples following the cessation of the activity. Dr. Zaldivar responded that either Miner does not clearly recall the test or is knowingly misrepresenting the procedure. (EX 12).

[7] Dr. Zaldivar testified that a CT scan was a useful diagnostic tool in determining the presence of coal workers' pneumoconiosis. (EX 9 at 18).

Treatment Records

Bluestone Health Center (EX 1)

Dr. Yoginder Yadav met with Miner for a follow up and a check up on March 20, 2003. Dr. Yadav also noted that Miner was getting a spirometry for the purpose of establishing a claim for Black Lung benefits. Dr. Yadav reported a coal mine employment history of 32 years. He also reported that Miner smoked two packs of cigarettes per day for an unspecified amount of time. Dr. Yadav based his conclusions primarily on an x-ray of Miner's chest as well as the pulmonary function studies that were conducted in conjunction with this follow up. He found radiographic evidence of both CWP and COPD. Dr. Yadav opined that the spirometry indicated the presence of a mild obstructive lung defect. He reported that the oxygen saturation was normal. Dr. Yadav also educated Miner on the benefits of smoking cessation, but he opined that Miner did not seem interested.

Bluefield Regional Medical Center (EX 2)

Miner had a consultation with Dr. Cuddalore P. Vasudevan on September 20, 2000. Miner was admitted due to a ten day period of "right upper quadrant abdominal pain." The reasons for a consultation were a high fever and an infiltrate found in the right lung on x-rays. After admission, Miner was diagnosed with leukocytosis. Dr. Vasudevan was directed to ascertain the cause of the leukocytosis and determine whether there was a connection to the right basal infiltrate. Miner denied any cough, congestion, "pleuritic type of chest pain," shortness of breath, swelling in the legs and hemoptysis. Dr. Vasudevan reported that Miner was a smoker and the time of the consultation and had smoked two packs a day "for many years." Upon examination, Dr. Vasudevan reported that "the breath sounds were normal, no adventitious sounds." Dr. Vasudevan also reviewed Miner's x-rays and CT scans and reported that the x-rays taken one month prior were "suspicious for a patchy infiltrate," whereas the x-rays taken in conjunction with the exam showed a "linear streaky atelectasis at the left lung base and a streaky atelectasis versus a blood vessel in the right lower lung zone of questionable significance, there is no infiltrate," which was supported by the CT scans. He attributed any changes to "gravitational changes." Dr. Vasudevan concluded that the fever and leukocytosis were most probably secondary to gallbladder disease.

Physician Opinions

Dr. D.L. Rasmussen (DX 13, CX 3)

The Department of Labor provides a full pulmonary evaluation to each Miner that files a claim for Black Lung benefits. Dr. Rasmussen, chosen by Miner to complete this evaluation, examined Miner on June 29, 2005. Dr. Rasmussen first recorded that Miner had progressively worsening shortness of breath for the six to seven years prior to the exam; a limited ability to climb a flight of stairs; a chronic productive cough; nightly wheezing and orthopnea. Miner reported being diagnosed with chronic bronchitis and remembered having pneumonia in 1963 or 1964. Dr. Rasmussen based his conclusions upon a 35 year coal mine employment history and

smoking history of 1.5 packs per day for 44 years. Upon examination Dr. Rasmussen found Miner's breaths sounds to be minimally reduced, however there were no rales, rhonchi or wheezes.

A chest x-ray, a blood gas study and a pulmonary function test were included in the evaluation. Dr. Rasmussen found radiographic evidence of CWP with a profusion of 1/0 throughout the lungs with s/s type opacities. Dr. Rasmussen reported that the ventilatory function studies indicated a slight obstructive impairment, that the single breath carbon monoxide diffusing capacity was moderately reduced and that the resting blood gas study results were within normal range. However, Dr. Rasmussen opined that the results of the exercise blood gas study indicated "marked impairment in oxygen transfer and he was at least minimally hypoxic." Based upon the exercise induced impairment, Dr. Rasmussen concluded that Miner did not have the "pulmonary capacity to perform his last regular coal mine job."

Dr. Rasmussen diagnosed both medical and legal pneumoconiosis. The diagnosis of medical coal workers' pneumoconiosis was based upon Miner's "significant history of exposure to coal mine dust" and "x-ray changes consistent with pneumoconiosis." Dr. Rasmussen concluded that Miner's lung disease was caused by both cigarette smoking and coal dust exposure. He opined that "both cause lung tissue destruction using similar cellular and biochemical mechanisms with resulting damage, which is indistinguishable by physical, physiologic or radiographic means. Although coal mine dust exposure may cause other tissue damage, which results in significant impairment in oxygen transfer absent or in excess of ventilatory impairment." (DX 13).

Dr. Rasmussen was deposed on December 7, 2006. (CX 3). He provided further explanation of his contention that coal dust and smoking cause similar damage.

> Emphysema, including centriacinar, panacinar, bullous[8], et cetera, all forms of emphysema, are caused by smoking, as well as coal mine dust exposure. The way the two dusts, smoke dust and coal mine dust, cause lung destruction is identical, the dusts are absorbed by the normal macrophages or scavenger cells, it's just part of the normal defense mechanisms, but in those susceptible individuals those cells give off a series of chemicals that start a whole cascade of cellular changes and enzymatic changes that end up dissolving – basically dissolving the lung tissue. Coal mine dust, in addition, causes interstitial fibrosis which is not caused by smoking. That fibrosis is most often present with the emphysema and some authorities believe that the fibrosis in the lung tissue prevents airway and alveolar collapse that would occur with emphysema alone and that, or course, is what's felt to be responsible for the frequent finding of impaired oxygen transfer either absent or in excess of airway obstruction. (CX 3 at 17)

[8] With reference to bullous emphysema, Dr. Rasmussen testified that "bullous emphysema is not a disease. Bullous changes can occur in any type of emphysema, including coal mine induced emphysema." (CX 3 at 20).

Dr. Rasmussen explained that emphysema alone does not typically cause Miner's pattern of impairment, namely impaired oxygen transfer in the absent or in excess of airway obstruction. He opined that typically with emphysema, "by the time you develop this much gas exchange, you would expect to have really considerable airway obstruction with reduction in FEV1." In response to Drs. Zaldivar's and Crisalli's claims that blood gas exchange is not significantly affected by CWP in the absence of ventilatory defect, Dr. Rasmussen stated that there has been no study to prove that contention. He references research studies and his personal experience as support against the contention put forth by the other physicians. Dr. Rasmussen reported having seen this impairment in miners who have never smoked, but also clarified that smoking accentuates the gas exchange impairment.

Dr. Rasmussen is a member of the American Boards of Internal Medicine, Forensic Examiners, Forensic Medicine, is a fellow in the American College of Forensic Examiners and a Senior Disability Analyst and Diplomate of the American Board of Disability Analysts. He was a chief medical officer at the Appalachian Coal Miners Research Coal Unit in 1966, the director of the Appalachian Pulmonary Laboratory in Beckley from 1973 to 1987, worked in the Division of Pulmonary Medicine at the Southern West Virginia Clinic from 1986 to the present. He was on numerous advisory boards and committees relating to Black Lung disease and mine safety. In the late 1960s, he received an honorable mention from the American Medical Association and an American Public Health Association Presidential Award for his work with Black Lung. He has published numerous journal articles about lung impairment in Coal Miners. (CX 3).

Dr. George Zaldivar (DX 14, EX 4)

Dr. Zaldivar examined Miner on both June 14, 2006 and August 10, 2005.[9] The August 10, 2005 evaluation consisted of a physical exam, an x-ray, a CT scan, arterial blood gas studies and pulmonary function tests.[10] Dr. Zaldivar also reviewed documents Miner submitted to the Department of Labor, specifically a questionnaire detailing his breathing difficulties and a description of his employment history, and an interrogatory completed by Miner on March 17, 2005 describing both the duties related to his work and his smoking history. Dr. Zaldivar recorded the history of Miner's present illness, Miner's past medical history, work history, personal history, social history and family history of illnesses. Miner informed Dr. Zaldivar that he had suffered from shortness of breath for at least three to four years; he could walk up two flights of stairs without stopping, but could not progress to the third flight; had experienced wheezing for three to five years; had been coughing up sputum in the mornings for four to five

[9] I find that the second examination exceeds the evidentiary limitations. Each party may submit two initial physician's opinions. In addition, supplemental reports may be admitted. A supplemental report is comprised of the physician's opinions in light of the rebuttal evidence. 20 C.F.R. §725.414(a)(3)(i) and (ii) (2001). Dr. Zaldivar's second report was based upon pulmonary function tests, arterial blood gas tests, an x-ray, a physical examination, and additional rebuttal evidence offered by Miner. The pulmonary function tests, blood gas studies, and the x-ray were all admitted into the record independently of his report. I do not find the actual physical examination to be necessary for Dr. Zaldivar to offer his opinion in light of further rebuttal evidence. Therefore, in my analysis, I will redact any comment pertaining specifically to June 14, 2006 physical examination of Miner.

[10] All of which were included in the record.

years and slept on one pillow. Dr. Zaldivar noted a coal mine employment history of 35 years and reported that Miner "began smoking at age 20 and now he smokes one and a half packs of cigarettes per day." Upon examination, Dr. Zaldivar found Miner's lungs to be "clear to auscultation without wheezes, crackles or rales."

Dr. Zaldivar completed the report associated with the above evaluation on September 5, 2005. Dr. Zaldivar concluded that Miner did not suffer from pneumoconiosis "nor any dust disease of the lungs," and that Miner's moderate exercise induced pulmonary impairment was caused by "bullous emphysema produced by his lifelong history of smoking." He also opined that from a pulmonary standpoint Miner "may not be able to perform his usual coal mining work, which according to [Miner] required heavy physical labor." Dr. Zaldivar reported that there was no radiographic evidence of CWP. The x-ray did not reveal any nodules of pneumoconiosis, which was supported by the findings of the CT scan, however the CT scan did show the presence of bullae. Dr. Zaldivar stated that "this bullae could not be attributed to coal workers' pneumoconiosis because coal workers' pneumoconiosis does not cause bullae." As support, he listed a number of journal articles which according to Dr. Zaldivar stated that coal dust exposure did not yield any evidence of "panlobular or bullous emphysema," but instead caused "centriacinar or focal emphysema which was directly related to the amount of dust retained within the lungs as shown by the macules and the presence of progressive massive fibrosis." Dr. Zaldivar stated that x-ray and CT scan evidence did not provide definitive answers regarding the presence of nodules in the lungs because it is possible that a pathologist may present contrary findings following inspection of the actual tissue. He did opine, however, that a CT scan "does determine … the amount of dust retained within the lungs." He reported that the medical literature indicated that "the greater the dust retention, the greater the changes radiographically and the greater the chances of damage produced by such dust retention."

Dr. Zaldivar noted that Miner's diffusion capacity was reduced, but opined that "the true reduction could not be determined because of his smoking habit which interferes with the diffusion, both through the high pressure of carbon monoxide in his blood and by the effects of smoking in the lungs."

Upon examination on June 14, 2006, Miner reported to Dr. Zaldivar that he smoked a pack of cigarettes per day and that he began smoking between the ages of 18 and 20. He also reported the same work history as was cited in the previous examination. Miner claimed to be able to ascend a flight of stairs, however he would be short of breath upon completion of the task. Dr. Zaldivar stated that Miner had increasing breathing difficulty for approximately four to five years. Miner had occasional wheezing in his lungs; however the wheezing was not related to any specific activity. Dr. Zaldivar noted that Miner slept on two pillows and did not have any chest pain or heart problem. Dr. Zaldivar noted Miner's family illnesses, personal and social history, and review of systems. Dr. Zaldivar found Miner's lungs to be clear to auscultation without wheezes, crackles or rales after the breathing test. Dr. Zaldivar's impressions based upon the examination were history of shortness of breath, normal examination of the lungs, and hypertension.

Dr. Zaldivar's original conclusions remain primarily intact following the most recent exam. Dr. Zaldivar explained that Miner was not able to exercise as long as he did on the prior

exam, however he attributed this change to either a lack of motivation or the use of a beta adrenergic blocker. Dr. Zaldivar diagnosed bullous emphysema, and noted that the bullae were much more easily seen in the most recent x-ray than on the previous x-ray, although they were easily seen on the CT scan taken in 2005. Dr. Zaldivar opined that Miner's pulmonary impairment will continue to worsen as Miner continues to smoke.

Dr. Zaldivar also had the opportunity to review Dr. Rasmussen's June 29, 2005 examination, pulmonary function studies and blood gas tests, the CT scan read by Dr. Scatarige on August 23, 2005, Dr. Wheeler's interpretation of the August 10, 2005 x-ray, and the interpretations of the June 29, 2005 x-ray by Drs. Rasmussen and Scatarige.[11] Dr. Zaldivar also had access to Dr. Vasudevan's treatment note from Bluefield Regional Medical Center and a statement regarding smoking history signed by Miner. Following his re-examination of Miner and review of additional medical records, Dr. Zaldivar concluded that his "opinion remains as previously given..." Dr. Zaldivar opined that Miner did not have CWP or any dust disease of the lungs, but rather suffered from emphysema caused by tobacco smoking. (EX 4).

Dr. Zaldivar was deposed on November 20, 2006. (EX 9) In his deposition, Dr. Zaldivar expanded upon the difference between the damage caused by smoking and coal dust exposure. Dr. Zaldivar opined that coal mine dust exposure and cigarette smoking do "not cause impairment in the same way." He explained that smoking causes an impairment through chemical damage to the lung that occurs because the "chemicals in the tobacco smoke are acting like… it's a foreign agent that is very irritating," thereby activating white cells and drawing them towards the lungs. These white cells congregate in the lungs and when they die they release elastases, which ultimately digest the lung tissue, and oxidases, which may damage the tissue locally, and in response the lungs create their own anti-oxidases and anti-proteases. Over time the lungs become less able to fight these chemical reactions and lung tissue is destroyed. In contrast, Dr. Zaldivar explained that CWP causes mechanical damage, whereby the white cells transport the inhaled dust particles up to the lymphatics, where they congregate around the respiratory bronchioles and cause localized fibrosis, which is considered a macule. "Distal to the fibrosis there is damage to the lung and that is the focal emphysema." (EX 9 at 16). Pertaining to the difference in the manifestation of emphysema caused by each factor, Dr. Zaldivar stated:

> Smoking causes centrilobular emphysema without any macules and it causes panlobular emphysema eventually where more and more units of the lungs are destroyed. And then you get into bullae which are large cavities within the lungs. That is smoking damage versus the coalworkers' pneumoconiosis damage which is a discreet damage called focal emphysema distal to the macule which causes the damage through particulate damage to the lungs. (EX 9 at 16).[12]

[11] Dr. Zaldivar also noted that Dr. Gaziano performed a quality reading on the June 29, 2005 x-ray and found the x-ray to be of good quality.

[12] When asked whether coal dust exposure can cause bullae, Dr. Zaldivar responded that simple CWP never causes bullae. However, he did explain that individuals with *progressive massive fibrosis* may have 'traction bullae' or 'traction emphysema', which occurs when a "big lesion retracts, it pulls lung with it and it tears the lung causing large holes, leaving large holes behind." (EX 9 at 31).

Dr. Zaldivar concluded that the radiographic evidence of emphysema was not accompanied by evidence of coal dust retention, and therefore "what we're looking at is emphysema… we're not looking at any dust damage in the lungs." (EX 9 at 19).[13]

Dr. Zaldivar testified that the ventilatory studies indicated the presence of a moderate airway obstruction, as indicated by the ratio of exhale volume in one second to the forced vital capacity. He then explained that, according to the American Thoracic Society, Miner's slightly abnormal ratio might not actually be abnormal because Miner's forced exhale volume is 100 percent of the predicted value. (EX 9 at 21). Dr. Zaldivar also opined that Miner's arterial blood gases results, collected at rest and during exercise, were within normal range. He notes a drop in PO2 during exercise, but explains that the drop was merely due to the "four millimeters of mercury rise". He did note that there was a significant drop in PO2 in blood gas studies conducted by Dr. Rasmussen. He opined that Miner's use of a beta blocker medication would not cause such a drop in PO2. Miner exercised for a longer period of time in Dr. Rasmussen's study. When asked to compare the two sets of results, Dr. Zaldivar looked at the printout of Dr. Rasmussen's report and stated "blood gases were not done throughout the entire test. I believe on this occasion he did two blood gases, one at the beginning and one at the end of the exercise."

Dr. Zaldivar disagreed with Dr. Rasmussen's conclusion that coal mine dust exposure can cause significant impairment in oxygen transfer in the absence of ventilatory impairment. Dr. Zaldivar testified that there is no literature supporting Dr. Rasmussen's contentions on this issue. He cited other studies whereby it was found that diffusing capacity was reduced only in smoking coal miners, indicating that such a reaction is not specific to coal miners because, according to Dr. Zaldivar, such a pattern is typical in smokers. He also referenced studies that showed a drop in diffusing capacity only in coal miners who had 'p' type densities in their chest x-rays. (EX 9at 33). Dr. Zaldivar concluded that "if the ventilatory study is normal and the individual has a drop in the PO2 with exercise, either they don't have coal workers' pneumoconiosis or dealing with a different disease, or you're having a problem with your results. Those are not the results of coal miners." (EX 9 at 34). He explained that a drop in PO2 in exercise in coal miners would be caused by destruction of the lungs that occurred as a result of airway obstruction brought on by the retention of coal dust.

Finally Dr. Zaldivar opined that, considering the nature of the position, Miner could return to his last job as a center control operator. Dr. Zaldivar testified that "all his lab works are good enough for him to perform any sort of work that he wishes to do. And certainly, his pulmonary capacity far exceeds what he is required to do on a daily basis, which is light moderate work." Dr. Zaldivar did concede that Miner might not be able to do the occasionally required heavy work, but attributes that inability to a lack of conditioning rather than any pulmonary impairment. (EX 9 at 30).

[13] Dr. Zaldivar opined that there is a lot of confusion regarding the connection between coal dust and emphysema; that originally coal dust was known to cause focal emphysema, but over time it seems as though some connect it to centrilobular. He explained that centrilobular emphysema was found in only in coal miners who also had smoking histories.

Dr. Zaldivar is board certified in Internal Medicine, Pulmonary Diseases and Sleep Disorder Medicine. He is a member of the American Thoracic Society, West Virginia Lung Association, the American College of Chest physicians, the West Virginia Thoracic Society, the European Respiratory Society and the American Medical Association. He has published articles pertaining to lung impairment and coal mining. He has been the speaker at numerous conferences and presentations, some of which pertain to occupational lung disease and respiratory care. He also attended numerous courses that dealt with pneumoconiosis. (EX 4).

Dr. Robert J. Crisalli (EX 3, EX 10)

Dr. Crisalli conducted a review of numerous medical records, all of which are included in the record, and drafted a report dated June 20, 2006. He reviewed the consultation by Dr. Vasudevan, the smoking history reported by Miner, Dr. Rasmussen's report, a CT scan and pulmonary function study performed by Dr. Zaldivar, a CT scan interpretation by Dr. Willis and Dr. Scatarige, and a chest x-ray interpreted by Dr. Wheeler and Dr. Zaldivar. The majority of Dr. Crisalli's report was a critique of Dr. Rasmussen's report.[14]

Pertaining to Dr. Rasmussen's June 29, 2005 report, Dr. Crisalli commented:

> "It is important to take the literature and the general conclusions described by the literature and apply it carefully to each specific case involving a patient. Each patient must be treated as an individual with physician's background knowledge of the general literature and the physician's experience. Though coal dust exposure can cause severe and disabling respiratory impairment, there is nothing in any of the articles cited by Dr. Rasmussen which states that any pulmonary function impairment in a coal miner is related to coal dust exposure or to coal workers' pneumoconiosis. There is nothing that states that all coal miners have pulmonary disease related to coal dust exposure."

Dr. Crisalli first opined that the CT scan did not support Dr. Rasmussen's x-ray interpretation. Dr. Crisalli explained that a CT scan is of a higher resolution, and so should be able to pick up nodules, however no nodules were found in the CT scan of Miner's chest.

Dr. Crisalli also addressed the presence of changes due to emphysema. Dr. Crisalli opined that "changes of bullous emphysema such as seen on the CT scan are associated with

[14] Dr. Willis' CT scan interpretation is not included in the record. In Harris v. Old Ben Coal Co., 23 B.L.R. 1-98 (2006)(en banc)(J. McGranery and J. Hall, concurring and dissenting), the Benefits Review Board indicated that when confronted with a medical opinion that contained evidence not admitted into the formal record, an administrative law judge may: a) exclude the report; b) redact the objectionable content; c) require a revised report; or, d) consider the physician's reliance on the inadmissible evidence in deciding the probative value of the report. Dr. Crisalli viewed a significant amount of evidence, including a CT scan with supposedly similar results. He did not specifically refer to Dr. Willis' interpretation in his report. I therefore find that Dr. Crisalli's reliance on this CT scan does not diminish the credibility of his opinion.

cigarette smoke exposure and are not associated with coal dust exposure." In support of this conclusion Dr. Crisalli explained that coal dust exposure typically causes focal emphysema, which "is not seen radiographically but can be seen under the microscope. This type of emphysema is microscopic and not nearly so extensive as to cause radiographic changes." Based upon the pulmonary function tests, Dr. Crisalli concluded that Miner has a "variable degree of obstruction to air flow and a diffusion study pattern which is characteristic for individuals with emphysema – that is, the diffusion capacity is low, the alveolar volume is normal or high, and the ratio between the diffusion capacity and the alveolar volume is reduced." As further support, Dr. Crisalli concluded that the blood gas study results showed oxygen saturation during exercise which he attributed entirely to emphysema and clarified that such results were in no way related to coal dust exposure. Dr. Crisalli concluded that "Dr. Rasmussen seems to believe that all lung disease in coal miners is somehow related to coal dust exposure. Dr. Rasmussen's assumptions and conclusions in this regard are not at all consistent with my experience as a pulmonary physician practicing in West Virginia for the past 29 years. It is not appropriate to attribute all lung disease in coal miners to the coal dust exposure and I have seen cases where treatable diseases were missed because the assumption was made that all lung disease in coal miners is related to coal dust exposure."

Dr. Crisalli opined that there is not a sufficient amount of objective evidence to support a diagnosis of CWP or "any chronic dust disease of the lungs." He does find Miner to be severely impaired as indicated by Miner's oxygen desaturation during exercise. He attributed the desaturation to bullous emphysema, which he stated is caused by smoking. Dr. Crisalli concluded that "[Miner's] case is one where he has significant exposure to coal dust and to cigarette smoke but where his lung disease is secondary only to the cigarette smoke and has nothing to do with coal dust exposure or coal workers pneumoconiosis." (EX 3).

Dr. Crisalli reviewed more recent medical evidence and stated his opinions in a deposition dated November 20, 2006. (EX 10). Dr. Crisalli opined that coal dust causes focal emphysema, which may be detected through pathology and "doesn't manifest itself on pulmonary functions studies… " and so is not expected to cause obstruction in airflow. (EX 10 at 11). Dr. Crisalli also stood by his opinion that coal dust exposure can not cause bullae because he had never encountered a miner with bullous emphysema that didn't smoke.

In response to Dr. Rasmussen's contention that the damage caused by smoking and coal dust is indistinguishable, Dr. Crisalli opined that the changes caused by each were different. As an example of the differences, he pointed to nodular densities and complicated pneumoconiosis, which are only caused by coal dust exposure and not smoking. There is a consensus between Drs. Crisalli and Zaldivar regarding whether coal dust exposure could cause defects in oxygen transfer without an obstruction to air flow. He mentioned Dr. Rasmussen's earlier studies on the topic, as did Dr. Zaldivar. He and Dr. Zaldivar also reported that Dr. Rasmussen's early studies were not supported by the medical literature. (EX 10 at 19). Dr. Crisalli completely ruled out coal dust exposure as a cause of the exercise induced desaturation because "the CT scans doesn't show any changes that [would be expected] with coal workers' pneumoconiosis, and they're very sensitive. The CT scans show changes of emphysema. The diffusing capacity is consistent with emphysema." (EX 10 at 20).

Dr. Crisalli is board certified in Internal Medicine with a certification in pulmonary diseases. He was the acting Chief of the Pulmonary Disease Section in the Department of Medicine at Marshall University School of Medicine in West Virginia from 1979 through 1983, the medical director of the Respiratory Therapy and Pulmonary Function Departments of Cabell-Huntington Hospital in West Virginia from 1982 though 1983, in the Veterans Administration Medical Center from 1977 through 1980, and Putnam General Hospital from 1984 through 1987. He was Chief of the Pulmonary Section of the Department of Medicine of Charleston Area Medical Center in West Virginia from 1988 through 1997. He was on the board of directors of the American Lung Association of West Virginia from 1987 through 1990. He has not published any journal articles pertaining to pneumoconiosis. (EX 3).

DISCUSSION

Because Miner filed his application for benefits after March 31, 1980, this claim shall be adjudicated under the regulations at 20 C.F.R. Part 718. Also, since this claim was filed after January 19, 2001, the regulations contained in 20 C.F.R. Part 718[15] as amended in 2001 are applicable. To establish entitlement to benefits under this part of the regulations, a claimant must prove by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. 20 C.F.R. §725.202(d); Anderson v. Valley Camp of Utah, Inc., 12 BLR 1-111, 1-112 (1989). In Director, OWCP v. Greenwich Collieries, et al., the U.S. Supreme Court stated that where the evidence is equally probative, the claimant necessarily fails to satisfy his burden of proving the existence of pneumoconiosis by a preponderance of the evidence. 114 S. Ct. 2251 (1994). The rulings of the United States Court of Appeals for the Fourth Circuit control in the adjudication of this case.

Pneumoconiosis

Under the Act, "'pneumoconiosis' means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 30 U.S.C. § 902(b). "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment. §718.201(a)(1-2).

Section 718.202(a) provides four methods for determining the existence of pneumoconiosis, (1) x-ray evidence; (2) biopsy or autopsy evidence; (3) if applicable, the

[15] All of the regulations cited in this decision are contained in Title 20 of the Code of Federal Regulations.

presumptions described in §§ 718.304, 718.305 or § 718.306 and (4) physician opinion evidence. Under § 718.202(a)(1), a finding of pneumoconiosis may be based upon x-ray evidence. In evaluating the x-ray evidence, I assign heightened weight to interpretations of physicians who qualify as either a board-certified radiologist or "B" reader. Dixon v. North Camp Coal Co., 8 BLR 1-344, 1-345 (1985). I assign greatest weight to interpretations of physicians with both of these qualifications.[16] Woodward v. Director, OWCP, 991 F.2d 314, 316 n.4 (6th Cir. 1993); Sheckler v. Clinchfield Coal Co., 7 BLR 1-128, 1-131 (1984).

This claim includes six interpretations of five x-rays. Two B-readers interpreted two different x-rays as positive for pneumoconiosis with a profusion of 1/0, which is the lowest qualifying profusion classification. The 1/0 classification means that although the physician felt that it was positive, the physician also considered that the film may be entirely negative. The four remaining interpretations were conducted by two dually qualified physicians.[17] Neither found evidence of CWP on the remaining x-rays. The physicians with the better qualifications failed to find pneumoconiosis. The lesser qualified physicians found pneumoconiosis, but also seriously considered the possibility that the film was negative. There is even conflict regarding the presence of pneumoconiosis in the two most recent x-rays. I find that the x-ray evidence does not establish the presence of pneumoconiosis.

Under § 718.202(a)(2), biopsy or autopsy evidence may be used to establish the presence of pneumoconiosis. Neither procedure was conducted in this case.

Under § 718.202(a)(3), a claimant may prove the existence of pneumoconiosis if one of the presumptions at §§ 718.304 to 718.306 applies. The presumptions at §§ 718.305 and 718.306 are inapplicable because they only apply to claims that were filed before January 1, 1982, and June 30, 1982, respectively. Section 718.304 requires x-ray, biopsy, or equivalent evidence of complicated pneumoconiosis. Complicated pneumoconiosis is diagnosed after a finding of an opacity greater than one centimeter is categorized as a type A, B or C. There is no mention of complicated pneumoconiosis in the medical evidence in the record; therefore I find this presumption to be inapplicable.

The final method by which the claimant can establish that he suffers from the disease is by well- reasoned, well-documented medical reports as per §718.202(a)(4). A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. Fields v. Island Creek Coal Co., 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination,

[16] I will refer to physicians with both of these qualifications as dually qualified physicians.

[17] Dr. Robinette interpreted an x-ray dated August **29**, 2006 as positive for pneumoconiosis. Employer offered Dr. Scatarige's interpretation of the August **28**, 2006 x-ray as rebuttal. This may be just a mistake as per the recorded dates of the x-ray. There is no circumstantial proof in the record to establish whether or not these interpretations are indeed of the same x-ray. Aside from the August interpretation by Scaterige, there are still three other negative interpretations by the most qualified physicians as compared to the two positive readings by the lesser qualified individuals. Therefore, even if I exclude Dr. Scatarige's interpretation of the August 28, 2006 x-ray, I would still find that the x-ray evidence does not establish the presence of CWP.

symptoms, and the patient's history. Hoffman v. B&G Construction Co., 8 B.L.R. 1-65 (1985); Hess v. Clinchfield Coal Co., 7 B.L.R. 1-295 (1984). A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. Fields, supra. Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder-of-fact to decide. Clark v. Karst-Robbins Coal Co., 12 B.L.R. 1-149 (1989)(en banc). Medical opinion evidence may establish either clinical or legal pneumoconiosis.

Clinical Pneumoconiosis

Only two physicians of record diagnosed clinical pneumoconiosis. Dr. Yoginder Tadav diagnosed pneumoconiosis based upon an x-ray that was not included in the record.[18] The Court and other physicians of record were unable to review and validate the underlying evidence. Therefore I find Dr. Tadav's opinion to be of little probative value with regards to the diagnosis of clinical pneumoconiosis.[19] Dr. Rasmussen diagnosed coal workers' pneumoconiosis and based his diagnosis upon his positive finding of pneumoconiosis in the June 29, 2005 x-ray, which is contrary to the finding of the Court. It is proper for the administrative law judge to accord less weight to a physician's opinion that is based on premises contrary to the judge's findings. Furgerson v. Jericol Mining, Inc., 22 B.L.R. 1-216 (2002)(en banc) (the ALJ 'did not reconcile physician's diagnosis of pneumoconiosis, based upon the positive x-ray and the miner's significant duration of coal dust exposure, with the fact that Dr. Baker's positive interpretation was reread as negative by a physician with superior qualifications; as a result, the Board directed that the ALJ 'address whether this rereading impacts the physician's opinion and his diagnosis of pneumoconiosis'). I grant Dr. Rasmussen's opinion regarding the diagnosis of CWP diminished weight.

Drs. Zaldivar and Crisalli both opined that there is no radiographic evidence of clinical pneumoconiosis. Their opinions are supported by the objective evidence included in this case. I found the overall x-ray evidence to be negative for CWP. Also, a CT scan, which is more sensitive than an x-ray, was interpreted as negative for clinical pneumoconiosis. I find that the preponderance of the probative medical evidence indicates that Miner does not suffer from clinical pneumoconiosis.

[18] In Harris v. Old Ben Coal Co., 23 B.L.R. 1-98 (2006)(en banc)(J. McGranery and J. Hall, concurring and dissenting), the Benefits Review Board indicated that when confronted with a medical opinion that contained evidence not admitted into the formal record, an administrative law judge may: a) exclude the report; b) redact the objectionable content; c) require a revised report; or, d) consider the physician's reliance on the inadmissible evidence in deciding the probative value of the report. I find that Dr. Tadav substantially relied on evidence not included in the record and therefore find his report to be of little probative value.

[19] The record also includes a treatment note from Dr. Vasudevan. This note is also based upon evidence not included in the record, nor does the note even mention the diagnosis of CWP. I find this treatment record to be of little probative value in the analysis of this issue.

Legal Pneumoconiosis

Virtually every physician of record recognized the presence of emphysema. A pulmonary disease may constitute statutory pneumoconiosis if it is significantly related to or aggravated by coal dust exposure in coal mine employment. The legal definition of pneumoconiosis is broad and may encompass more respiratory or pulmonary conditions than those specifically, clinically diagnosed in a medical opinion. For example, a physician may conclude that the miner suffers from asthma that is related to his coal dust exposure. Although the physician did not specifically state that the miner suffered from pneumoconiosis or black lung disease, the respiratory condition that he diagnoses is related to coal dust exposure and, therefore, is supportive of a finding of legal pneumoconiosis. The question in this case is whether Miner's emphysema arose from coal dust exposure, and therefore qualifies as legal pneumoconiosis.

Drs. Zaldivar and Crisalli opine that Miner's emphysema and resulting impairment are not related to coal dust exposure. Dr. Rasmussen contends that both coal dust exposure and cigarette smoking can cause identical forms of damage, namely emphysema, and Miner's specific pattern of impairment is indicative of the presence of fibrosis caused by coal dust exposure. All three physicians agree that coal dust exposure can lead to emphysema. However, Dr. Rasmussen claims that coal dust can cause any form of emphysema, whereas Drs. Zaldivar and Crisalli both state that coal dust typically causes focal emphysema and centrilobular emphysema is caused primarily by cigarette smoking. Drs. Zaldivar and Crisalli provide very persuasive explanations regarding the formation of bullae and the exclusion of coal dust as a cause of such a phenomenon.[20] Dr. Rasmussen provides a contradictory and general explanation contending that any "dust" (pertaining to both coal dust and cigarette smoke) inhaled into the lungs will cause a chain of self defense mechanisms that will ultimately cause damage to the lung tissue. According to Dr. Rasmussen's contentions, any Miner with emphysema and substantial coal dust exposure would have legal pneumoconiosis. Dr. Crisalli commented that there is no literature to support the conclusion that "any pulmonary function impairment in a coal miner is related to coal dust exposure or coal workers' pneumoconiosis." If such were the case, there would be no need for any Miner to establish the presence of pneumoconiosis, rather all that would be necessary would be proof of a pulmonary impairment. No such presumption exists.

In his deposition, however, Dr. Rasmussen did address the specific physiologic findings in this case. All three physicians agreed that Miner displayed an impairment in blood gas transfer without the presence of a significant pulmonary obstruction. Dr. Rasmussen opined that such a pattern is indicative of damage caused by coal dust. He testified that "fibrosis is most often present with the emphysema and some authorities believe that the fibrosis in the lung tissue prevents airway and alveolar collapse that would occur with emphysema alone and that… is what's felt to be responsible for the frequent finding of impaired oxygen transfer either absent or in excess of airway obstruction." I find this statement to be equivocal and lacking support from the objective medical evidence included in this case. I do not find the observation that *some authorities believe or feel* that fibrosis may be the cause of a similar impairment to be strong

[20] Dr. Zaldivar clarified that bullae may be caused by coal dust exposure, but only when there is progressive massive fibrosis, which is not present in this case.

enough to carry Miner's burden of proof. Also, the overall evidence in this case does not support a finding of fibrosis in the lungs.[21] Dr. Rasmussen does provide a few references for his contentions. Drs. Zaldivar and Cristalli provide references for their opposing view points. I am unable to determine the credibility of these references as none of the journal articles were included in the record. I will therefore defer to the qualifications of the physicians involved. Although Dr. Rasmussen has significant experience specifically pertaining to pneumoconiosis and pulmonary disease, he lacks board certification in pulmonary disease. Dr. Crisalli is board certified in pulmonary disease, but has less experience relevant to pneumoconiosis. Dr. Zaldivar possesses both direct experience with pneumoconiosis and a board certification in pulmonary disease. I find that Dr. Zaldivar is better qualified than Drs. Rasmussen and Crisalli.

Drs. Zaldivar and Crisalli provide both well-documented and well-reasoned opinions. Some of Dr. Rasmussen's statements are too general in nature. A medical opinion based upon generalities, rather than specifically focusing upon the miner's condition, may be rejected. Knizer v. Bethlehem Mines Corp., 8 B.L.R. 1-5 (1985). Other conclusions made by Dr. Rasmussen, although more specific to Miner's case, are not based on the objective evidence of record. Dr. Rasmussen is also less qualified than Dr. Zaldivar. I therefore rely on the consensus between Drs. Zaldivar and Crisalli and find that preponderance of the probative medical opinion evidence does not establish the presence of clinical or legal pneumoconiosis.

Miner did not establish the presence of clinical or legal pneumoconiosis; therefore the other three elements of entitlement are moot. The preponderance of the evidence does not support a finding of pneumoconiosis, therefore Miner's claim for benefits must be denied.

ORDER

L.R.P.'s claim for benefits under the Act is hereby **DENIED.**

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LARRY W. PRICE
Administrative Law Judge

[21] Dr. Zaldivar did comment that x-rays and CT scans are not infallible in the detection of nodules in the lungs. However, in this case, there is no other evidence of fibrosis to corroborate Dr. Rasmussen's theory that fibrosis is the cause of Miner's specific pattern of impairment.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).